Reprinted for the Author from the BRITISH MEDICAL JOURNAL,
July 22nd, 1893.

CLINICAL REMARKS

ON

INTRAVASCULAR COAGULATION AND PULMONARY THROMBOSIS.

BY SIR JOSEPH FAYRER, K.C.S.I., M.D., F.R.S.

ABOUT thirty years ago I began to call attention in India to the formation of clots and fibrinous coagula in the heart and great vessels as a source of danger, and frequently of death, not only to persons who were suffering from various forms of disease, but also to those who had sustained injury or undergone surgical operations, in many cases where they seemed to be otherwise progressing favourably. I remarked the apparent influence of dyscrasia and cachexia depending on malarial infection and splenic disease and on conditions arising out of various other forms of toxæmia, in producing the state of blood in which this intravascular clotting is apt to occur.

I was unable then to give more than a general account of or to offer more than somewhat vague suggestions as to, the nature of the cause, but the light which has been thrown on the subject by the recent researches of pathologists in Britain and on the Continent, and which were so well expounded in the Goulstonian Lectures of 1893 by Dr. Halliburton, F.R.S.—his own contributions to the subject being of great interest and value—has now furnished a solution which goes far to explain that which has hitherto been obscure, and offers also reasonable ground for hope that a death cause often so terrible in its sudden onset and rapidly fatal termination may be in some instances mitigated, if not altogether prevented, and may prove amenable to remedies which chemico-physiological investigation has demonstrated to possess the power of controlling this abnormal tendency to intravascular coagulation.

I venture in the following communication to recur to observations made by me many years ago, and to relate briefly a few cases which illustrate the pathological conditions referred to. I would repeat my opinion that among the conditions which tend to determine this dangerous state of the blood, that which is so prevalent in tropical and malarial climates, more especially when any splenic complication exists, is a fertile cause of clotting, and that it frequently induces death by pulmonary thrombosis, not only in cholera, fever, and other diseases, but after surgical operations, not infrequently even where the progress of the sufferer appeared otherwise to be favourable.

The very lucid explanation given by Dr. Halliburton in his recent lectures and the account of his researches, as well as those of Wooldridge, Wright, W. L. Dickinson, E. H. Hankin, Green, Ringer, Sainsbury, Shore, Hardy, Schmidt, Hammarsten, Virchow, Brücke, Arthus, Pagès, Pekelharing, Grusbach, Löwit, Lilienfeld, Daremberg, into the nature and pathological causes of the processes by which it is brought about appear most conclusive, and are a striking example of the great practical value of researches conducted in the patient, persevering, and scientific spirit characteristic of those recorded and acknowledged by Dr. Halliburton in the lectures above referred to.

I am glad to find that the impression made on me by former experience of this disease, to wit, that the depraved state of the blood in malarial and splenic cachexia, in which the leucocythæmic condition is productive of danger by its tendency to cause intravascular clotting, thus receives explanation and confirmation from these pathologists, and I feel that to them are due grateful thanks for the light thrown on an important and hitherto obscure cause of suffering.

When listening with deep interest to Dr. Halliburton's lectures, I heard him refer to a particular case of pulmonary artery thrombosis which occurred in this country; my thoughts immediately reverted to many such cases which had happened in my own experience. I have therefore offered the following contributions to the subject, and I believe that Dr. Halliburton will find in them illustrations of the views which his most valuable researches have developed, and I would repeat what I have said on other occasions, that I believe intravascular coagulation with pulmonary thrombosis to be a more frequent cause of death, in this country as well as in India and the tropics, than is generally supposed. I would by no means be understood to imply that this is due only to malarial influences. I merely wish to point out that I believe that a condition of the blood which may be induced by many causes is more prone to occur in climates where such influences exist.

I have little doubt that physicians and surgeons, looking back over former experiences, will recall cases where death has occurred owing to this condition, in which necropsy has revealed the presence of a clot in the pulmonary artery as the only cause to which the fatal termination may be assigned.

The above remarks refer chiefly to clotting in the venous system, but the arterial side of the circulation is by no means exempt from it, and this I have described in the paper, "Gangrene from Arterial Embolism," p. 131 of *Clinical and Pathological Observations in India*, where I have given illustrations of it as manifested in gangrene or other lesions resulting from plugging of the main arteries of the limbs or other parts of the body.

In this paper reference is made chiefly to thrombosis of the pulmonary artery, and the following examples are given where the right ventricle, the pulmonary artery, and its extreme branches have been found, after death, to be plugged by a firm, white, fibrinous clot, which had rapidly proved fatal.

Case I. *Ligature of the Femoral Artery for Elephantiasis of the Leg; Death from Fibrinous Coagula in the Pulmonary Artery.*—A Bengali, aged 30, was admitted on February 22nd, 1865, with elephantiasis of the right leg of seven years' duration; otherwise he was healthy looking, with no symptoms of disease elsewhere. The femoral artery was tied at 9 A.M. on the 25th, near the apex of Scarpa's triangle. He did well until March 4th.

when he had slight fever; this recurred at intervals of a day or two, without, however, affecting the foot until the 14th, when he felt a burning sensation in both legs. On the 13th pyæmic symptoms had set in, with a rapid pulse and hurried breathing. These symptoms were worse on the 14th, and he died on that day at 2 P.M. The wound had looked healthy all the way through.

Post-mortem Examination.—The liver and spleen were healthy; the kidneys were infiltrated with pus. The iliac veins appeared healthy, but the right contained a clot. The lungs contained numerous patches of dead tissue, one as large as a small orange. There was also purulent effusion into the cavities of the pleuræ. The pericardium contained a quantity of yellow serum. The right ventricle was completely filled with a firm decolorised clot, which had moulded itself in the cavity and extended far into the pulmonary arteries. There was no sign in the condition of the femoral artery of any pathological change that would have given rise to mischief. There was no phlebitis in the femoral vein.

CASE II. *Operation for Elephantiasis; Pulmonary Thrombosis; Death.*—A Mahomedan, male, aged 50, admitted January 26th, 1866, with elephantiasis of scrotum. On February 1st tumour was removed. There was much bleeding from the wounded vessels. The pulse was very weak; the wound looked healthy throughout. On the 8th he had pain in the right hypochondrium, with hurried breathing, and died on the 9th.

Post mortem, there were whitish decolorised clots in the right auricle and ventricle of the heart, extending into the pulmonary artery and all its minute ramifications in the lung. Pulmonary veins were plugged with similar clots, which extended into their branches and filled the left auricle. Other organs were healthy.

CASE III. *Operation for Elephantiasis; Pulmonary Thrombosis; Death.*—J. H., aged 45, admitted on August 24th, 1866, with scrotal tumour of two years' duration. Tumour removed on September 3rd; lost much blood; temperature rose; pulse small and weak; became delirious; breathing hurried; died on the 10th.

Post mortem, a firm, whitish clot in the upper vena cava was found extending into the right auricle, which was nearly filled by it. Clot extended into the right ventricle, through the tricuspid valve, and thence into the pulmonary artery and all its branches. Pulmonary veins filled with similar clots, which passed into their ramifications and filled up the left auricle; the left ventricle was empty. There was a clot in the aorta, which was adherent to the valves, and ended in a free extremity beyond the origin of the left subclavian artery, where it was floating loose within the calibre of the vessel. The lungs were blanched, and seemed rather shrunken; other organs healthy, except the kidneys, which were pale and flabby.

CASE IV. *Opening of a Deep Sinus; Pulmonary Thrombosis; Death.*—A Hindu, male, aged 38, was admitted on March 27th, 1867, suffering from a deep sinus running under the gluteal muscles. A counter opening was made. Had fever for the next three or four days. An incision below the great trochanter gave exit to some fœtid pus and gas. Fever ceased on 31st. Pulse weak; breathing very hurried; discharge fœtid and thin; incision enlarged; died on April 2nd.

Post-mortem Examination.—The left lung was found much congested; right lung contained a small tuberculous cavity at its apex; no pyæmic patches, no pleuritic effusion. Right auricle and ventricle of heart completely stuffed with firm fibrinous coagula, extending into the pulmonary arteries. Left lobe of liver much enlarged; its substance soft and fatty; spleen much enlarged; kidneys flabby, and their capsules easily detached. The sinus ran under the gluteus maximus, and had no communication with the bone. The tissues in the neighbourhood were in a state of decomposition.

The formation of fibrinous coagula in the cavities of the heart, or in the great vessels, is a condition which we know is liable to occur in the later stages of numerous diseases, and probably it is the actual cause of death in many, being itself due to the altered condition of the blood; perhaps to some extent to the diminished vitality of the tubes and cavities in which the blood circulates, and to altered or defective nerve force. It is not only to its occurrence under these circumstances that I now allude, but to the fact that it is a danger to be apprehended after any severe—indeed not always severe—operation or injury, and may occur independently of those signs which indicate the toxæmic state of the blood we so frequently see in hospital patients in large cities, where not only are the hygienic conditions defective,

but the people themselves are anæmic and wanting in vital energy. It is right, therefore, that this source of danger should be borne in mind, and that in any case where a tendency to exhaustion or anæmia exists, particular attention should be paid to the diet, and administration of such remedies as may tend to counteract the disordered condition, which we may fairly assume to exist when these symptoms appear. In the preparation of patients for an operation, as well as in their treatment afterwards, it should be borne in mind that iron and nutrients should be freely given to improve the condition of the blood. Hyposulphites, according to Polli; alkalics, according to Richardson and others, are indicated; the former to limit the development of poison germs, the latter to obviate the tendency in the blood to clot.[1] Above all, plenty of fresh air, to oxygenate the blood and tissues. Such, no doubt, are the measures of a therapeutic and dietetic nature from which we may hope to derive benefit. In many cases, the supervention of this obstructed condition of the right side of the heart is the precursor of speedy death, but not necessarily always so; and doubtless a certain amount of clotting is frequently recovered from, or its effects are seen in a secondary form in the changes which occur in the lungs as the result of the capillary embolism to which its *débris* may give rise. In those cases wherein it proves fatal, the end is speedily brought about by the rapid and sudden withdrawal of blood from the pulmonic circulation, syncope or cardiac apnœa closing the scene.

The earliest symptoms of weakness, failing pulse, coldness of the extremities, with rapid action of the heart, pallor or lividity and hurried respiration, should receive due attention; and in some cases I believe that, provided no other changes have occurred in the viscera, as a result of septicæmia, the timely administration of remedies such as I have indicated may avert the danger and save life.

Case No. IV is an example of the conditions I have been describing. It is true there were *post-mortem* evidences of visceral changes of a chronic nature, but they were principally such as tend to induce the condition in which fibrinous clots are most likely to form, by interfering with the due elaboration and development of healthy blood; for if there be any one condition more than another unfavourable to such development, it is that disordered state of the blood-making organs so frequently found in malarious countries. It is, I believe, only right that in estimating the respective influences of the various causes by which life may be endangered or lost, after surgical operation, that we should take into consideration the malarious state of the climate, and its effects; and whilst I admit that much of the mortality may be due to intrinsic causes, I feel more strongly than ever the paramount necessity of having healthy, well-ventilated hospitals.

CASE V. *Amputation of Arm; Pulmonary Thrombosis; Death.*—A Hindu, aged 40, admitted May 6th, 1867, with lacerated wound on inner aspect of left elbow-joint, inflicted with a pickaxe. Pus formed in the wound, he did not do well, had rigors and fever, indications of pyæmia appeared, so amputation was performed through the lower third of the humerus. Fever continued; complained of pain in the left side of the chest; respiration hurried; temperature high, more rigors; died June 5th.

Post-mortem Examination.—The lungs were found to be œdematous and emphysematous, pale, but right lung congested posteriorly. Left lung had three pyæmic patches of dead lung tissue, saturated with puriform

[1] Drs. Halliburton and Wright suggest other remedies for the purpose.

sanies. Left pleural cavity contained about 5 ounces of serous fluid, with flakes of lymph. Upper lobe of left lung covered with thick layer of lymph. Firm decolorised clot in right auricle and ventricle, extending far into pulmonary artery. Liver pale and slightly enlarged; spleen considerably enlarged. Bone contained pus infiltrated throughout medullary cavity. Other organs healthy. Death occurred from pyæmia, but immediate cause was cardiac thrombosis, interrupting pulmonary circulation.

CASE VI. *Injury to the Scalp; Pulmonary Thrombosis; Death.*—A native, aged 25, admitted May 13th, 1867, for severe injury to scalp. Bone in right frontal region laid bare to extent of 4 by 2 inches. Pericranium removed, but bone itself not injured. Did well for first week. After that had much pain in head, rigors, fever, sweating, inclination to vomit, rapid pulse, and high temperature. Trephine applied over centre of denuded portion of bone, which had dull and dry appearance. On reaching the diploë, a large quantity of fœtid pus was evacuated; no pus between bone and dura mater. No improvement resulted; breathing became more hurried; became unconscious; died the same evening.

Post-mortem Examination.—The frontal bone was found in complete state of diffused osteomyelitis, diploë being infiltrated with fœtid pus. Large quantity of thin pus under dura mater, diffused over surface of that side of the brain, but none between the skull and the dura mater. There was one small pyæmic patch on the surface of the middle lobe of the right lung, and another on the left lung. The right auricle and ventricle were plugged with firm, adherent, fibrinous clots, extending into the pulmonary artery. Liver, kidneys, and spleen healthy. The cause of death in this case was undoubtedly interrupted pulmonary circulation, as proved by the clots in the heart and blanched and emphysematous condition of portions of lungs. Small pyæmic patches in lungs were quite incipient.

CASE VII. *Amputation of the Leg; Pulmonary Thrombosis; Death.*—A Hindu, aged 21, admitted June 24th, 1867; right knee injured by an agricultural instrument. Wound was found to communicate freely with the joint; sanious discharge; increased much, became puriform and fœtid. Amputation performed at the lower third of the thigh on July 2nd. Did not do well. Symptoms of toxæmia appeared and progressed rapidly, and on July 4th amputation at hip-joint was performed. Pulse and temperature high; delirium; rapid breathing; no hæmorrhage from stump. Died on July 5th, breathing having become very hurried.

Post-mortem Examination.—The right ventricle contained firm, flattened, and adherent decolorised clot, extending far into ramifications of pulmonary artery. Right auricle stuffed with firm white clot, supplemented by a more recent one. Lungs blanched, some portions being quite exsanguine, others containing a little blood; both emphysematous and œdematous. No structural change in any of the viscera. Nothing remarkable in stump; medulla infiltrated with pus. The immediate cause of death appears to have been coagula. Absence of structural change in the viscera points to the fact that recovery might have taken place had coagula not formed.

CASE VIII. *Injury to the Rectum and Bladder; Pulmonary Thrombosis; Death.*—Conductor H. was admitted on December 16th, 1868, suffering from effects of an accident which had happened eight months previously. He had slipped down a hill a few feet on to a stake, which pierced the right gluteal region and penetrated the bladder. He was in great pain for the first day or two, but after then until July 11th he did well. On that day a great deal of blood came away through the wound. The bladder and urethra became very irritable, and he suffered much from tenesmus. Under treatment the symptoms improved, and when he was admitted, on December 16th, he seemed in fair general health, but complained of an increased desire to pass water, with much pain at the neck of the bladder. Calculus was detected, and on January 7th the lateral operation was performed and a friable calculus removed. Had frequent attacks of fever and diarrhœa, and twice incisions were made in the inguinal canal and deep-seated pus evacuated. On February 9th, when apparently convalescing, he went home for change of air. He returned on February 27th much worse; had had severe rigors and fever; pulse feeble and rapid; respiration gasping and hurried. On February 20th the breathing became much more rapid and gasping, and intense cardiac apnœa preceded death.

Post-mortem Examination.—The lungs were found to be much congested posteriorly. Firm decolorised clots in the right cavities of the heart, extending far into the ramifications of the pulmonary artery. Liver enlarged; contained very numerous pyæmic patches of the size of peas. Spleen congested and softened; kidneys somewhat congested.

This was an exceedingly interesting as well as instructive case from the beginning. His recovery from so grave an accident, in the first place, was very remarkable. A stake driven through the gluteal region and rectum into the bladder

might well have proved rapidly fatal, and the result wonderfully illustrates the reparative power inherent in the constitution of a man in the vigour of health. He had so far recovered in about seven months as to be able partly to resume his duty. The formation of the calculus may be accounted for, no doubt, by the condition of the bladder injured by the wound; a nucleus having formed, determined by the roughened and irregular surface of that part of the bladder where the fistula opened, and perhaps by the entry of some hard substance from the rectum, the concretion rapidly gathered round it and produced the calculus detected on his admission. No part of the stake with which he was injured could be found in the bladder, though carefully looked for. His subsequent condition was not less remarkable. There can be little doubt, I think, that the train of unfavourable events which preceded his death were mainly due to the influence of malarious poisoning. The fever that supervened after the operation was most probably of malarious origin, and the blood, already thus poisoned, was more readily affected by the toxic conditions excited by the operation. The fibrinous coagula in the heart, which were the immediate cause of death, were no doubt due to the same causes. That the absorption of septic matter in this case took place mainly through the portal circulation is indicated by the state of the liver, which was studded with local deaths of tissue; and the enlarged spleen tends to support the theory that malarious poisoning was much concerned in inducing a state of the blood generally which finally accelerated the fatal result by determining the formation of fibrinous coagula in the right cavities of the heart.

CASE IX. *Contusion of the Leg; Pulmonary Thrombosis; Death.*—A Bengali, male, aged 20, was admitted on April 15th, 1870, with contusion on the right leg. Had been suffering from malarious fever and enlarged spleen for five months previously. Leg and knee were œdematous. The bruised portion, just below the knee-joint, looked as though it were becoming gangrenous. Temperature low; pulse small and feeble. Remained in this state for two days. Respiration began to be hurried on the 18th; became more gasping and hurried; died at midnight on the 20th in a state of extreme cardiac apnœa.

Post-mortem Examination.—The spleen was found to be much enlarged. The lungs were somewhat congested hypostatically, and a portion of the lower lobe of the right lung was consolidated. In the heart a firm fibrinous coagulum was found extending from the right auricle, where it was reddish, into the ventricle, where it was straw-coloured, and thence firm and fibrinous into the pulmonary artery and its minute subdivisions. A similar one was found in the left cavities, extending into the aorta. The integument was gangrenous for several inches down the leg. The knee-joint was not compromised; and, on being laid open, its structures were found to be normal.

There was certainly not sufficient in this case, in the mere gangrene of the integument of the leg, to account for death in an ordinary individual; but in a person suffering from malarial blood poisoning and enlargement of the spleen, it was more than sufficient. Probably in no condition of the disease is the formation of fibrinous coagula more likely to occur, on the least disturbance, than in splenic or malarious cachexia. In a marked case, such as this, where the spleen was four or five times its natural size, it may be said that there is nothing remarkable in its termination; for do we not see such almost daily in the cases of cancrum oris, sloughing ulceration, and necrosis, that are unhappily so common in Bengal, and probably in other localities wherever malarial influences are rife? The imperfect condition of the blood-making organs, and the impoverished character of the blood they elaborate, are amply demonstrated in the leucocythæmia.

and in the great tendency to disintegration and death of the soft tissues and bones. The evil results of thrombosis, when it occurs in the arterial circulation, are seen in the limbs or other parts of the body, which become gangrenous, or, still worse, in the multiple deaths of portions of the viscera, which are so frequently observed in the so-called pyæmic conditions generally met with after wounds and injuries, though by no means infrequently, idiopathically.

I have repeatedly called attention to the subject as one of great importance in a surgical point of view, for it is not only in cases where an enlarged spleen renders almost any operation impossible, and causes almost any wound to prove fatal, but in many others, whether of wound or injury, where there is no obvious disease of the spleen, and where all seems to be doing well, that this may and often does supervene, and rapidly carry off the sufferer. The condition is one most common in exhaustive diseases, and it is, no doubt, often one of the latest pathological phenomena manifested by the moribund, but it is more than this, for, as I have said, it may set in where there is no appearance of exhaustion, when repair and nutrition are going on satisfactorily, and within twenty-four or forty-eight hours carry off the patient, whose body presents no solution of the cause of death beyond a firm, white, adherent clot in the right auricle or ventricle, or it may be just at the ostium of the pulmonary artery, which is indeed the janua vitæ.

This condition taking place in the right side of the heart or in the pulmonary artery is one of the dangers that the subject of a surgical operation, wound, or injury has to encounter, and not merely as the last act of a series of pathological processes, the result of exhaustive or prolonged disease, but as an original and dangerous consequence of some blood change. What the nature of this change may be I am uncertain; it may perhaps arise out of the presence of matters retained in the blood that should have ministered to the nutrition of the part removed in cases of amputation or ablation of parts of the body, a condition somewhat analogous perhaps to the retention in the body of a secretion that should have been eliminated; or, in cases where no removal of parts has occurred, it may be due to some disturbed condition in which the blood is prone to coagulation. I have a strong suspicion that climate is not without influence in originating this dangerous state. In Bengal all are more or less under the influence of malaria; it is true, happily, that in a large majority of persons its effects are not generally perceptible.

I do not regard this as altogether due to a malarious condition of the blood; I know that, although it may not have been noticed as a result of surgical operations, Dr. Richardson long ago pointed out its tendency to occur in exhaustive diseases; but I cannot help thinking that a pathological state capable of producing so many important changes as malarial infection does, may have something to do with this one also.

Case x. *Catheterism; Urethral Fever; Pulmonary Thrombosis; Death.*—An English sailor, aged 39, applied for relief from retention of urine on June 20th, 1870. He said he had suffered from slight stricture for the last five or six years. A catheter passed and urine drawn off without difficulty. Returned in the afternoon, very ill, in great distress about the stomach; pain across abdomen or lower part of the chest. Had passed some urine tinged with blood after his return home. On May 21st the pain seemed to localise itself in the right hypochondriac region, and

there was excessive tenderness on pressure over the liver. Breathing hurried. No urine had been passed since admission; dulness on percussion over the base of the right lung; breathing hurried and gasping; distress increased, and he complained that something was choking him. At about 10.30 he passed water, but no improvement resulted, and he died at midnight "in a sudden fit of gasping." Struggle for breath was most distressing to witness.

Post-mortem Examination.—The lungs were hypostatically congested, the right most so; one portion of the middle lobe was solidified; lower lobe was also hepatised. There were one or two very small patches of tuberculous deposit in the apices of the lungs; with this exception they were healthy and crepitant. Heart was normal, but its cavities contained firm, adherent, fibrinous clots; that in the left side extended from the auricle into the ventricle and into the aorta for about 3 inches; that in the right was larger, and extended from the auricle through the ventricle into the finer ramifications of the pulmonary artery; the pleuræ were normal. The kidneys were congested; the other viscera and peritoneum were healthy. The bladder was somewhat thickened, and the urethra was slightly strictured in front of the bulb; there was no wound and no false passage.

Case x is very instructive. A man in the prime of life, in good health, with the exception of a slight stricture, a steady, intelligent, and temperate person, suffers from retention of urine, due to a slight excess a day or two previously. He is relieved at once by the passage of a No. 8 catheter. He returns home, feels chilly, has rigors, rapidly followed by fever and delirium; he passes urine tinged with blood after his return home. The fever is attended with intense restlessness and distress; severe pain in the right hypochondriac region and pit of the stomach follows. Rapid, deep, and gasping breathing, with the greatest præcordial distress, increase and go on getting worse and worse, until the patient dies in great agony of breathlessness about forty hours after the catheter had been passed. His intellect was perfect to the last; urine was secreted and voided not long before death. It is clear, therefore, that the symptoms were neither due to uræmic or eholæmic poisoning, nor to any cerebral disorder. There was no exhaustive disease. Air entered the chest freely, and his voice was natural to the last. There was neither heart disease, peritonitis, nor other acute inflammation. What, then, was the cause of death?

The *post-mortem* examination revealed some congestion at the base of each lung. The pleuræ were healthy, and the pericardium contained a small quantity of fluid. None of these conditions were sufficient to cause death. The abdominal viscera were not absolutely healthy, for the liver was slightly enlarged, and the kidneys were congested. The bladder, prostate, and the tissues about its neck were generally healthy.

The bladder was somewhat thickened in consequence of a slight stricture situated just in front of the bulb of the urethra. Through this an instrument had been passed. There was nothing in the abdomen to account for death; but on opening the heart, it was evident that the formation of coagula had destroyed life. They were firm, decolorised, and adherent, and on the right side not only obstructed the auricular, ventricular, and arterial openings, but extended far into the subdivisions of the pulmonary arteries, ramifying like the branches of a tree.

I have repeatedly noticed this condition as a cause of death, but I have never seen a more striking example than this. I have before mentioned my suspicions that malaria has much to do with inducing the condition of blood in which this coagulation occurs. In this case, the simple act of passing a catheter through a slightly strictured urethra was the

exciting cause. The patient cannot be said to have been, strictly speaking, in a healthy condition, although he appeared in perfect health, and no lesion was discovered that would account for death. Sufficient, however, existed to suggest how, with the addition of the shock of the operation, and the consequent urethral fever, the blood change and consequent cardiac thrombosis were brought about.

Something was said about his having recently suffered from intermittent fever, but he had none of the appearances of malarial cachexia. But no one in Bengal can be said to be exempt, especially at that season of the year; and I am convinced that this malarious influence, which affects all more or less, is in persons of irritable constitution, and especially in those suffering from stricture, a predisposing cause of that dangerous condition—urethral fever—in which, under certain conditions, fibrinous coagulation is likely to occur.

It has long been known that death may occur rapidly from this cause in puerperal patients, and that in diphtheria, croup, cholera, and other exhaustive diseases, it is not by any means infrequent; and in such cases, where the blood must necessarily be in an altered condition, and the muscular fibre of the heart weak, although the precise nature of the change may be unknown, it is not difficult to understand why the fatal result should occur. But in cases such as the preceding, and in others where fatal cardiac thrombosis has supervened after surgical operations, when the patient appeared to be doing well, the explanation is more difficult. That it occurs in persons in whom there are no obvious indications of cardiac disease is the more reason why, in a hot and exhausting climate, it should take place in those who are the subjects of fatty degeneration, or when the heart is loaded with fat externally, or where there are either pleuritic or pericarditic effusions, or the ventricles are dilated, and the muscular fibre atrophied. In such persons, indeed, the presence of fibrinous concretion may not be needed to bring about the fatal apnœa. The temperature of a night in May or June in a tropical climate such as Calcutta, the shock or after-effects of an operation, may be sufficient; and the patient, after a short and distressing struggle of breathlessness, perishes.

CASE XI.—*Malarial Poisoning; Pulmonary Thrombosis; Death.*—On August 6th, 1871, I saw a case of supposed irreducible hernia with symptoms of strangulation. The patient was a young Englishman, aged 28, with a swelling in the left groin below Poupart's ligament, painful on pressure, with general abdominal tenderness; smaller swelling in the other groin, less painful. Was feverish, and had been so during the night, with severe pain, nausea, and vomiting. He was easier in the morning, but was sent to the hospital. At about 2 P.M. the symptoms of the night recurred, and at about 4 P.M., breathing, which had been hurried before, became more so; cold sweats covered his body, and all the indications of collapse set in; breathing was painfully gasping and hurried; face and lips dusky and livid; he was conscious to the last. Sank soon after 4 P.M.

Post-mortem Examination.—It was seen that there was no hernia; the swellings in the groins consisted of thickened areolar tissue around a cluster of indurated glands, and had been present for months if not years. The lungs were intensely congested, especially at the back, and contained numerous patches of pulmonary apoplexy throughout their structure; they were œdematous, and the bronchial mucous membrane was deeply congested and covered with frothy mucus. The heart was normal and firmly contracted; both sides contained small, firm, decolorised clots extending into the aorta and pulmonary artery; all else was normal.

This is a very interesting as well as a somewhat obscure case. It is an example of the rapidly fatal effect sometimes produced at this season of malarious poisoning, the weather at the time being hot, damp, and replete with malarious in-

fluences. The symptoms were such as not unnaturally, considering the state of the groins, suggested strangulated hernia; indeed so much so that I was tempted to cut down on the swelling to remove all doubt. But when I saw him after removal to the hopital, the collapse was so complete that I did not do so. Death was due to the pulmonary engorgement accelerated by the formation of fibrinous coagula in the right side of the heart. The overwhelming action of the malarious poison can only account for the rapidly fatal phenomena. The patient was a healthy and temperate man, his organs were sound, and he had not been ill for months before the day on which this illness commenced. A certain amount of enlargement of the scrotum, combined with hydrocele, together with swelling in the groins, suggested the existence of an elephantoid taint; although in other respects he was free from disease.

Case xii. *Carbuncle; Pulmonary Thrombosis; Death.*—A Lascar aged 28, was admitted October 20th, 1871, with a carbuncular swelling on the right cheek, involving lips and lower eyelids. The corresponding mucous membrane lining the cheek was covered with a croupous-looking exudation. Disease attributed to slight cut whilst shaving. General health much impaired by malarious enlargement of liver and spleen. Small incisions were made in cheek and lip, from which purulent matter found exit. Patch of exudation removed from the cheek. On October 22nd, pulse feeble, no fever, respiration very hurried; delirium; more punctures made. On October 23rd he was worse; breathing excessively gasping and hurried; died at 3 P.M.

Post-mortem Examination.—The lungs were hypostatically congested. No pyæmic changes. There was found a firm fibrinous clot in right ventricle, extending from the auriculo-ventricular opening into the finer ramifications of the pulmonary arteries. Smaller clot occupied the left ventricle, extending into the aorta. Liver and spleen both enlarged; other viscera appeared normal.

Case xiii. *Carbuncle; Pulmonary Thrombosis; Death.*—A native (male), aged 27, was admitted January 8th, 1872, with carbuncular swelling of the left cheek, the living membrane of lip on that side being affected with a diphtheritic exudation. Commenced eight days previously with a small pimple at the entrance of the nostril. Health impaired by malarious enlargement of the spleen. Incisions made, but only thin sanious fluid evacuated. On January 9th there was slight fever. Next day he was worse, respiration being very rapid and hurried. Sank and died of apnœa.

Post-mortem Examination.—The muscular structure of the heart was pale and flabby. A large coagulum, partially decolorised, occupied the right auricle and ventricle, and extended into the pulmonary artery. Dark deposits, varying in size from a walnut to a pea, were found scattered through the substance of the lungs. Malarial enlargement of liver; spleen also somewhat enlarged.

Case xiv. *Injury to the Head: Pulmonary Thrombosis; Death.*—An English boy, aged 4½, fell on July 15th and cut the back of his head slightly. It bled freely, and his mother applied a piece of sticking plaster. For a week after he was considered to be in his usual health and spirits. In the night, about a week afterwards, he was feverish, restless, and sick. In the morning still feverish and light headed, with a peculiar twitching of the muscles. When examined, the wound was found to be deep, the scalp round it was swollen, œdematous, boggy, but not red; there was no positive evidence of pus. He rapidly became worse; was violently convulsed; became silent; respiration became excessively hurried; was quite unconscious; pupils widely dilated; died at 4 P.M.

Post-mortem Examination.—The scalp was reflected. All round the wound it was infiltrated with a dark red serous, and probably partially purulent, fluid. This occupied an area of an inch in each direction; beyond this and down to the neck it was infiltrated with turbid serum. All this part of the scalp was œdematous. The wound itself was healthy, and reached nearly, not quite, to the pericranium, which was unwounded. The pericranium was detached from the bone for about a square inch, corresponding to the wound. The bone was bare but did not appear dead. The bones of the cranium were healthy; the section revealed no suppuration in the cancellated textures. The longitudinal and other sinuses were distended with blood, and contained coagula. The brain was much congested on the surface; the vessels between the convolutions were engorged; the surface of the base, especially over the wound, was slightly ecchymosed under the arachnoid. There was no meningitis, nothing suggestive of tuberculosis. The brain substance, the ventricles, and the membranes were healthy; lungs pallid, almost blanched; heart firmly

contracted; pericardium natural; right auricle and ventricle contained a peculiarly tough decolorised fibrinous clot, firmly wedged in the auriculo-ventricular opening; did not extend into pulmonary vessels; pleuræ were natural, viscera healthy.

Case xv. *Splenic Cachexia; Pulmonary Thrombosis; Death.*—A young officer, about 23 or 24, returned from India after suffering severely from repeated attacks of malarial fever, complicated with excessive enlargement of the spleen, and a profound state of splenic cachexia. Under the influence of proper care and the use of quinine and sulphate of iron he was making slow, but apparently satisfactory, progress. His condition of debility had been complicated by occurrence of hurried respiration with a weak and irregular pulse. He had been enjoined to observe perfect rest and quiet, and to avoid all attempts at muscular exertion. One day, being attracted by a sudden noise in the street, he rose from his bed, hurried to the window, and made an effort either to close or open it. He was immediately attacked with hurried and gasping respiration, staggered back to his bed, on to which he fell prostate. The symptoms continued, and within a period of an hour or so he died. To a clot already existing a fresh accretion had taken place, which rapidly brought his life to a close.

Case xvi. *Cholera; Pulmonary Thrombosis; Death.*—A civil officer of high rank, middle-aged, was attacked with cholera in Calcutta. He passed into a state of collapse, and his life was in great peril for some hours. By aid of treatment and careful nursing he was recovering. The vomiting and purging had entirely ceased; the urinary secretion, which had been entirely suspended, was restored. He was regarded as being in a favourable condition, with every prospect of recovery, when symptoms of pulmonary obstruction supervened. His respiration became gasping and hurried, and he died in a few hours. A clot had formed in his pulmonary artery, which was the cause of death. There can be little doubt that had this untoward condition not supervened, his recovery might have been complete.

The cases here recorded proved fatal, but death does not always result; many recover, some quickly, some after protracted suffering. But recently I have had the opportunity of observing in consultation with Surgeon-Lieutenant-Colonel Platt a case in which clotting had occurred in both the arterial and venous circulation. Both femoral arteries had been compromised, whilst life had been several times in extreme peril from symptoms of coagula in the right side of the heart. Recovery is now slowly, but apparently satisfactorily, progressing.

The cases noted sufficiently illustrate the dangerous and fatal effects of cardiac and pulmonary thrombosis. Others have occurred in my experience of which I have kept the notes, but it seems needless to multiply them. It is to be noted that however much or little these cases may be referable to malarial causes, septic conditions were then very rife in Indian hospitals.